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Editorial Comment

PHILADELPHIA PENNSYLVANIA PLAN PROGRESS

TWELVE County Medical Societies in Pennsylvania now have Tuberculosis Committees appointed, and many more County Societies are in process of forming such Tuberculosis Committees, to cooperate with the State Medical Society Tuberculosis Committee to secure the very necessary reforms in the State Tuberculosis Sanatoriums and State Tuberculosis Clinics.

It is hoped that in the State Sanatoriums it will soon be possible to *Work* the *Sanatorium Beds More,* and the *Patients Less;* to attract to the sanatorium the type of doctors and nurses who wish to make the treatment of tuberculosis their lifework, through giving them proper salaries, a rising pay scale, and security in their jobs under civil service. We hope to be able to give the patients their meals at 9 a. m., 1 p. m., and 6 p. m., instead of at 7 a. m., 11:30 a. m., and 4:15 p. m., as is done at present.

These and many more necessary changes will all cost money. We cannot do with $1.40 to $1.60 per day maintenance per patient, that which costs properly functioning tuberculosis institutions $3.00 to $3.40 per day.

The State Assembly will supply the necessary funds—they have always been willing to meet the necessary tuberculosis expenditures, but all of the monies that they have been appropriating have not been spent on Tuberculosis!

Have you ever heard of "Reallocation of Funds?" Bureaucrats sometimes by this means spend money on their hobbies which never would have won the support of the Legislature, at the expense of the Tuberculosis Clinics and Tuberculosis Sanatoriums.

It is the duty of Organized Medicine through its State and County Tuberculosis Committees to watch over these matters, and see to the correction of faults which could exist only through the apathy and lack of organized, directed knowledge *within* the Medical Profession.

F. W. B.

FRANKNESS IN TUBERCULOSIS

IN some respects tuberculosis is unique in that it calls for the exhibition of considerable frankness on the part of the attending physician. In many other conditions this attitude may not be wise, and at times actually dangerous. It has often been stated that you can frighten a pneumonia patient to death. Certainly, it is the best policy, when possible, to refrain from letting a patient know that he has pneumonia, until he is well along in his convalescence. It is best to offer as an explanation for his illness the assuring diagnosis of flu, acute bronchitis or congestion of the lungs. It is only under exceptional circumstances, and to the exceptional patient, that you can freely render the

verdict—cancer. It is unnecessary and probably inhuman to sweep away all hope, to fill the soul with dread and despair. Such wreckage usually follows in the wake of this formidable diagnosis. The fear and subsequent invalidism following the pronouncement of heart trouble, even though it be only the detection of a systolic blow at the apex; are well known. In these and many other conditions, tact and judgment are urgently demanded in order to properly treat the patient, without provoking the morbid responses that follow when such discretions are not observed.

In tuberculosis, it is different. In this disease, it is wisdom to be frank with your patient, to take him into your confidence, to discuss the case openly and intelligently. The cooperation of the patient is demanded in tuberculosis as in no other disease; and it is only by this cooperation that any reasonable hopes for recovery can be anticipated. To give this cooperation, the patient must have a clear and thorough understanding of his own illness. Of course, tact and judgment are to be observed here; and brutal frankness in cases with a grave prognosis is to be decried. Always be cheerful and optimistic where possible, even if you have to "bend over backwards" a bit to be so.

The doctor sees the tuberculous patient relatively infrequently; but the patient lives daily with his illness, and must be informed so as to know the warning signals. So much in the proper handling of the case depends upon the recognition and interpretation of these signals. There is a saying—"What you don't know, won't hurt you." The brilliant exception to this rule is in tuberculosis. Here, it does hurt not to know.

It has been the experience of the writer that where the family forces the true nature of the disease to be withheld from the patient suffering from tuberculosis, that an unfavorable outcome always results. To refuse to take a case where this is demanded would be justifiable. It is most interesting, and at times somewhat amazing, to witness patients discussing their cavity, their pneumothorax, their "phrenic" as something apart—yet so intimately and vitally their own.

Therefore, in taking care of your patients with tuberculosis, let frankness be the order of the day. C. H. H.

TUBERCULOSIS CONTROL

OUR efforts toward tuberculosis control have increased year by year, especially since 1905. After all these years of intensive study and work, what measures are now proven to have had a real part in our declining death rate?

In a recent study of this problem by Drolet, we find many interesting facts. He says "With declining cases and deaths, a common observation, there can be little question but that, between improved conditions of life and increasing provisions for the sick, some control of the infection is obviously being achieved. On the other hand, is definite progress being made in the treatment of the disease now that there is available in many places a number of hospital beds sufficient to receive most of the active cases who seek public care, and, of late, new and radical forms of surgical treatment are being used in an increasingly greater portion of the patients? What effect, if any, have these facilities and measures had upon the case fatality rate in tuberculosis?"

He had made an intensive study of the case and fatality rate of many of the larger cities in this country and abroad, the studies extending from the year 1915 up to 1936. In all of these cities, the case rate has gradually decreased, while the fatality rate has actually increased in some instances. For example, in New York City, in 1915, there were 22,141 cases reported with 8,825 deaths, or 40 per cent fatality rate. While in 1935 there were only 8,796 cases reported with 3,968 deaths or a 45 per cent fatality rate, an actual increase of 5 per cent in 1935 over 1915.

In practically all of the other cities studied, with the exception of Detroit, he found a large decrease in the number of cases reported but the fatality rate remained practically the same in 1935 as it was in 1915.

His studies, therefore, clearly show a definite decline, both in the death rate and number of cases; the percentage in each instance being practically the same. But the fatality rate remains about the same at present as in 1915.

From these studies, it would seem that there

is little conclusive evidence that institutional, or modern treatment, has had any marked influence upon the community case fatality rates from tuberculosis, still it is obvious that by segregating an increasingly large proportion of open cases there are preventative values which are in a great part responsible, along with the improvement of living and environmental conditions for the acknowledged decrease of tuberculosis.

It would seem from these studies that the most valuable contribution down through the years toward tuberculosis control has been the gradually increased numbers of available beds where open cases can be isolated. It has been our opinion that the segregation of open cases is the most important factor in tuberculosis control. C. M. H.

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TEXAS TUBERCULOSIS ASSOCIATION

HILTON HOTEL

April 15 and 16, 1938

FRIDAY, APRIL 15th

Registration—8:30 a. m.

9:30—*Social Work and Administrative Section.*

"Dawn"—A skit depicting the part of the public health nurse in the Tuberculosis program—College of Mines Players.
Director—Mrs. Myrtle Ball.
Technical Adviser — Miss Norine Baumgarten, R. N., County Health Dept.

Symposium on Programs for the Control of Tuberculosis in Texas:

Program of Taylor County Tuberculosis Association — Miss Lena Wilson, Executive Secretary.

Program of Beaumont Tuberculosis Association—Mrs. Mildred Cobb, Executive Secty.

Program of Houston Anti-Tuberculosis League—Miss Emmeline Renis, Executive Secretary.

Statistical Summary of the Activities of all Associations Affiliated with The Texas Tuberculosis Association—Miss Pansy Nichols, Executive Secretary.

Program of the State Tuberculosis Sanatorium—Dr. J. B. McKnight, Supt.

Program of the State Department of Health—Dr. Howard Smith, Director of the Division of Tuberculosis.

Opportunities for Co-operation Between Voluntary and Official Health Agencies—Dr. C. St. C. Guild, National Tuberculosis Association.

LUNCHEON — 12:30

Use of Social Security Funds in the Public Health Program—Dr. F. W. Kratz, United States Public Health Service.

Rehabilitation of the Tuberculous—Mr. Holland Hudson, National Tuberculosis Assn.

2:30—*Medical Section.*

Bone and Joint Tuberculosis in Children—Dr. G. W. N. Eggers, Superintendent, Children's Hospital, Galveston.

Tuberculin Testing—Dr. J. A. Myers, Minneapolis, Minnesota, President, National Tuberculosis Association.

Finding Tuberculosis in Infants—Dr. Elva A. Wright, Houston.

Paper by Dr. Donato G. Alarcon, Mexico City, Director del Sanatoria para Tuberculosos de la Beneficencia Publica, Huipulco, D. F. Subject to be announced.

The Tuberculosis Problem in the State of Chihuahua—Dr. Jesus Olmos M., Servicios Sanitarios Coordinados en el Estado, Chihuahua, Mexico.

DINNER — 7:30

Address—Dr. H. F. Carman, President, Texas Tuberculosis Association.

Accomplishments of the Past Thirty Years in the Program for the Control of Tuberculosis—Dr. J. A. Myers, President, National Tuberculosis Association.

Influence of Research in Tuberculosis on the Practice of Internal Medicine—Dr. Henry Winans, Dallas.

SATURDAY, APRIL 16th

9:30—*Medical Section.*

Surgical Management of Bilateral Cavernous Tuberculosis—Dr. J. Emerson Daily, Houston.

Tuberculous Enteritis—Dr. James Gorman, El Paso.

Laryngeal Tuberculosis — Doctors Schuster and Schuster, El Paso.

Gas and Oil Hazards in Refineries and Oil Fields and Their Relation to Respiratory Diseases — Dr. L. J. Moorman, Oklahoma City.

SEAL SALE LUNCHEON — 12:30

2:00—*X-ray Clinic.*

3:00—*Business Meeting*—Members and Board of Directors.

Atmospheric Pollution in Relation to Tuberculosis*

SIR PENDRILL VARRIER-JONES, M.A. (Cantab.), F.R.C.P. (Lond.)**
Cambridge, England

AT the Memorial Service held in honour of the late Lord Moynihan Blake's "Jerusalem" was sung. Not until then did I realize that those immortal, oft-sung lines contain what is in all probability a reference to the smoke nuisance:—

"And did the countenance Divine
"Shine forth upon our *clouded* hills
"And was Jerusalem builded here
"Among those *dark satanic* mills."

This led me to enquire into the age of the smoke menace; and in a book published in 1914 by Dr. Guy Hinsdale[1] I found a very interesting footnote. It appears that six hundred years ago King Edward the First was so moved by it that he made the use of "sea-coal" punishable by death. This violent enactment did not last, however, and smoke had again become a notable nuisance in the reign of Queen Elizabeth. The seventeenth century diarist, John Evelyn[2], described at considerable length the smoke of London in his time; and his contemporary, Charles the Second, did actually adopt what Hinsdale calls "repressive measures" in London. In the early nineteenth century London was colloquially known as "the Smoke"; and in the second year of the present century, Hinsdale states, a singer brought an action against the city of St. Louis, Missouri, and its chief smoke inspector, on the ground that "owing to the additional presence of smoke, suffocating gases and acid, the health of the complainant was injured."

Like the poor, therefore, and like disease, it appears that smoke has been always with us. Of late years, however, thanks to the efforts of the Smoke Abatement Society and kindred organizations in other countries, scientific thought has been concentrated upon the estimation of the effects of smoke upon the public health, and with greater and greater accuracy we are able to assess its cost.

As if we did not know it already, a popular song not long ago assured us that "Smoke Gets in Your Eyes." If that were all it got into I should not be here to-day; but as it also gets into your lungs I have been asked to speak upon atmospheric pollution in relation to tuberculosis.

With your permission I will deal with this question in three sections. First I will briefly review existing literature upon the subject. Next, I will indicate my conclusions; and then I will submit to you the results of some recent work at Papworth which make my subject far more complex, and many times more important, than it would have been even six months ago.

Smoke and coal dust in the air compel us, willy-nilly, to inhale quantities of tar, oil, and volatile products of combustion. Of that there is no doubt; but as to the effect thus produced there are several apparently conflicting opinions.

Ascher[3] suggests that the increase of smoke in industrial towns must have some bearing upon the increase in diseases of the upper respiratory tract, because, according to his investigations acute pulmonary diseases are more frequent in industrial towns than they are in the country. This sounds reasonable, since Gautier[4] claims that nine thousand kilograms of H_2SO_3 and HCl descend upon Paris from the air every year. I have not precisely corresponding figures for London; but I am told that ten million pounds worth of coal similarly descends upon London, via our myriad smoky chimneys. When it is remembered that H_2SO_3 destroys chlorophyll even in a dilution of one in a million, and when one considers the effect of air-borne acids upon our public buildings, it would seem strange indeed if the delicate membranes of the respiratory tract should not be affected in an even more serious degree. But does tuberculosis result?

The answer appears to be in the negative.

* Read before the Eighth Annual Conference of the National Smoke Abatement Society, October 16, 1936, Science Museum, South Kensington, England.

** Director, Papworth Village Settlement, Cambridge, England.

Smoke has not, to my knowledge, ever been proved guilty of causing tuberculosis by means of its effect upon the respiratory tract. On the contrary, the experiments on rabbits carried out by Gross[5] indicated that smoke was probably not harmful; and Joetten and Arnoldi[6] who tested the relative irritation of various dusts, found that soot was the least irritating. More important still for us, Dr. Franz Ickert[7] quotes Claissé and Josué, who gave animals soot to inhale and found that no fibrosis developed until tubercle bacilli were mixed with the soot.

Other evidence points in the same direction. Dr. Georg Rosenfeld[8] of Breslau, has indeed been led to make a colloidal solution designed to counteract tuberculosis by increasing the coal content of the lung; and Dr. H. G. Obermeyer[9] states that "some authorities have gone so far as to claim that carbon deposits in the lungs offer a 'kind of immunity against this type of infection.'" This view is supported by Professor Lyle Cummins[10] who has expressed the opinion that there is a factor in coal mines which tends to neutralize the danger of tuberculosis.

Let us now consider the reaction of coal miners to tuberculosis. These men are exposed to far more coal dust than anyone else; and if it is air-borne coal which causes tuberculosis we shall expect to find among them a very high mortality rate. But this is just what we do not find. Even after ten years' exposure coal miners contract tuberculosis only rarely, *so long as* they are exposed to coal dust alone, and not a mixture of coal and silica. Bohme[11] found that 1.4 per cent of coal dust in the tissues did not produce any proliferation of connective tissue, whereas as little as 0.7 per cent of SiO_2 is always associated with silicosis in a very severe form. Heymann and Freudenberg[12] find that the incidence of tuberculosis among coal miners is below the average. Only 3.8 per cent of them contract the disease, while 10.3 per cent of stone and ore miners do so. Finally, Arnold[13] after studying the statistics of a whole century, also found that the tuberculosis mortality of coal miners was below the average tuberculosis mortality of the whole population.

What are we to conclude? I think we must admit that the inhalation of smoke arising from the combustion of coal has no causative effect in relation to tuberculosis, but may, and almost certainly does, have a protective effect. Whether that protection is worth having, whether indeed we would not rather be without it, is a matter which I will discuss later. Smoke palls must, with almost equal certainty, favour the spread of the disease. Tonney and de Young[14] say that, in Baltimore, Shrader, Coblentz and others have reported a loss, due largely to smoke, of 50 per cent in solar ultra violet light as determined by actinic methods, and I cannot believe that such a reduction is without effect upon the metabolism generally and the calcium metabolism in particular. The state of calcium deficiency must weaken the resistance to tuberculosis; but this weakening may to some extent be compensated by the mysterious protective factor already mentioned. That fogs resulting from atmospheric pollution accelerate the deaths of tuberculous persons is clear from the evidence of Dr. Veitch Clark[15] and his staff at Manchester; but weighing all the factors I am of the opinion that we cannot justly and wholly blame smoke for the tuberculosis death-roll.

So far, in using the word tuberculosis I have done so in its hitherto generally accepted sense. I have treated it as though it were a self-contained disease, quite unlike and unrelated to any other. But in this the correct view? Our recent work at the Papworth Laboratory powerfully suggests that it is not; but that, on the contrary, tuberculosis has a very near, and very unpleasant, relation indeed.

Before going any further into this, let us now for a moment consider the association between smoke and cancer which has been noted by so many authorities. Here all the evidence seems to point one way, which is so unusual that it must surely be conclusive. Of particular interest is a paper by Dr. Jerome Meyers[16] of the New York Department of Health. He quotes C. E. Green's[17] investigations which show that epithelioma is "very prevalent among chimney sweeps, who show the highest rate for any occupation, also among gardeners or farmers who use soot," and that "cancer is known to be frequent among workers in aniline dyes, paraffin, and gas pitch, brewers and metal workers who use sulphuric acid." Bertillon[18] working geographically discovered the areas in France

wherein cancer incidence was greatest; and Green[17], pursuing this clue, found that the highest cancer death rates occurred in the coal-burning areas, and the lowest in the wood-burning areas. Dr. Meyers also refers to the work of Kennaway[19], whose work on the cancer-producing factor in tar is well known, and who concludes his work on cancer produced by gas-works tar, lignite tar and shale oil by saying that "the attempts made as yet to find the cancer-producing substance among the well-known constituents of coal-tar have given wholly negative results. It is not unlikely that this substance is a compound, as yet unknown, which is unstable and present in amounts so small as those of the vitamins in foods; as in the case of some hormones its identification may be long delayed even when very concentrated preparations may be obtained."

Dr. Meyers' own conclusions are very interesting. Early in his paper he says that while it is not claimed that smoke accounts for all cases of cancer "it is probably one of many causes, or one manifestation through biochemical irritation of some great underlying cause that is as yet beyond our ken"; and at the end, after describing the interesting and illuminating Staten Island experiment, he includes in his summary a reference to a "cancerogenic factor liberated by combustion."

IS THAT FACTOR, I WONDER, LINKED WITH THE PROTECTIVE FACTOR NOTED BY LYLE CUMMINS IN RELATION TO TUBERCULOSIS?

I do not say that it is; I only ask. And I do not ask only out of curiosity, but because an affirmative answer would not be inconsistent with the suggestion, which I am about to submit, that so far from cancer and tuberculosis being two separate diseases there is new evidence to show that they are so very closely related that it is unprofitable any longer to investigate one without reference to the other.

Some time ago I invited one of our research staff at Papworth, Dr. D. Barron Cruickshank, to investigate tuberculosis from a biological standpoint. He did so; and the result of his study is enshrined in a very remarkable paper which is about to be published by the Sims Memorial Laboratory, Papworth, under the title "Tuberculosis, Cancer and Zinc."

The first section of this paper is a survey of existing literature on zinc metabolism. It is a fairly exhaustive survey, traversing a very wide range of research. Reference is made to the facts that coal ash may contain 2½ per cent zinc; and there is another point of special relevance to what follows. It is shown how zinc is ingested; and it is also shown that water, milk and other substances corrode, and thereby become to some extent impregnated with, the zinc constituent in galvanized iron. Thus milk, for example, of which incidentally zinc is an initial constituent, becomes further impregnated with zinc if it be kept for any length of time in a galvanized iron container. I want you to bear that in mind.

In the next section Dr. Cruickshank draws attention to the very considerable decline in chlorosis, better known as anaemia. This decline is admittedly associated with the availability of iron in medicinal form. This association has been observed, and is not now questioned.

Dr. Cruickshank passes on to consider the decline in the tuberculosis mortality, and asks himself whether there is any factor, hitherto unobserved, which may be associated with this decline, in the same way that available iron is associated with the decline in chlorosis. He draws attention to the depressing but inescapable fact that the decline in the tuberculosis mortality began years before sanatorium treatment was instituted; and to the still more humiliating, but equally inescapable, fact that the discovery of Koch's bacillus, and the extensive panoply of treatment based upon that discovery, have not in the least accelerated that decline. Yet the decline is real, and progressive. It must evidently be due to something. It cannot just happen. What, then, is the cause?

It is notable that the rate of decline is associated with the rise in the purchasing power of wages, and the consequently enhanced consumption of foodstuffs, including milk. This is reasonable enough, for we have always held that good food and plenty of milk assisted the mechanism of resistance to tuberculosis. But why should the peak of the tuberculosis mortality — according to Carl Pearson — be reached in 1838? Why not 1708, or 1886? We do not know: no one knows. But Dr. Cruickshank points out that it was in 1837 that Dr. H. W. Cranford obtained a

patent for the first galvanised iron container; and that since then galvanised iron has been in increasingly general use. It is not unreasonable, I think, to suggest that galvanised iron may have increased the availability of zinc and this instituted the decline in tuberculosis, in just the same way that the availability of iron has admittedly led tc the decline in chlorosis.

Ceasing for the moment to be statistical Dr. Cruickshank then becomes geographical: and he finds that in countries where zinc is present in the soil there is in general high resistance to tuberculosis, but that in areas where zinc is absent resistance to tuberculosis —of the human, as opposed to the bovine type—is low. Becoming botanical, he shows how zinciferous soil influences the synthesis of plants; and then, becoming zoological, he traces the effect of zinc transmitted via the flora and the fauna. Thus he finds that rats, goats and dogs, animals notoriously refractory to tuberculosis infection, all originated in zinciferous districts; whilst peculiarly susceptible animals, such as guinea pigs and apes, derive from territories where no zinc is present in the soil.

Becoming medical, he observes that peoples indigenous to non-zinciferous areas have little nor no resistance to tuberculosis; whereas zinc workers, who are inhaling zinc particles all their working lives, while often afflicted by respiratory diseases such as bronchitis and pneumonia, have higher resistance to tuberculosis than any other class of worker.

Thus, gentlemen, you see that the significance of zinc is considerable. In quantity it is inconsiderable; but it appears to be essential not only to the growth and development of many plants and animals but also to the organisation of resistance to tuberculosis. That, too, I ask you to bear in mind.

Next, Dr. Cruickshank turns to the astonishing fact observed by Dr. Cherry[20] namely that cancer has increased in such an exact ratio to the decline in tuberculosis that the sum of the cancer-tuberculosis mortality has remained constant for more than eighty years. Since 1851 cancer and tuberculosis have together accounted for 20 per cent of the total deaths after the age of 25 each year, accidental deaths excluded. As tuberculosis has fallen, so has cancer increased: the combined toll remaining constant at 20 per cent of the total mortality.

That alone would suggest that there must be some relation between the two diseases: the more especially as there is no such statistical relation between any two other diseases.

Dr. Cruickshank then enquires into the possibility that zinc deficiency may open the door to tuberculosis while zinc excess may lead to cancer. In this connection he notes that it has already been demonstrated that in cancerous tissue the zinc content is relatively high, and that the higher the zinc content the greater the degree of malignancy. Further, that zinc concentrates in the nucleus which controls cell development; so that it may be regarded as either a causative, or a protective, factor. Since, however the zinc workers, whose resistance to tuberculosis is so marked, suffer a higher cancer mortality rate than any other workers, he is forced to the conclusion that whilst zinc may be a protective against tuberculosis, it may be a causative factor in relation to cancer. In this belief he is sustained by the consideration that the animals from zinciferous areas are resistant to tuberculosis, while animals from non-zinciferous areas are resistant to cancer. Take, for example, the rat, the rabbit and the guinea pig. In order of susceptibility to tuberculosis the guinea-pig comes first, the rabbit next and the rat last. In order of susceptibility to cancer the exact converse is true. The rat is most susceptible; the rabbit next and the guinea-pig last. Thus the thread of zinc seems to run through, and connect, both diseases.

At this point Dr. Cruickshank seeks to discover the mechanism which will answer a very puzzling question. Since, as Dr. Cherry's statistics show, cancer and tuberculosis are *exactly* antagonistic, the question arises: Can tuberculosis cause cancer if cancer is itself a resistance to tuberculosis?

In search of this mechanism Dr. Cruickshank considered the group of intestinal infections. In these, as you know, the pathogenic bacilli are lysed by the action of non-pathogenic bacteriophages. The condition of the patient depends entirely upon the victory of the one over the other. If the pathogenic bacilli are victorious, the patient dies. If the bacteriophages win, then the bacilli are lysed

and the patient recovers.

Hitherto, as Dr. Cruickshank points out, it has been perhaps too readily assumed that all bacteriophages are non-pathogenic. Indeed, it is only recently that research workers have begun to realise that there may be as many species of phage as there are of bacteria. Dr. Cruickshank felt that there was a strong probability that some of these phages might be pathogenic; and he then applied this conception to the question before him. He argued that if the mechanism of defence in tuberculosis could be shown to correspond with the mechanism of defence in the coli diseases, the existence of a phage, probably identical with the lytic factor already demonstrated by Steenken[22], could be presumed; and further that if this phage were *pathogenic,* it could account, as could no other factor, for the exact relation between the cancer-tuberculosis mortality figures.

From this, Dr. Cruickshank has been bold enough to suggest—and in my view to substantiate—the idea that at the moment when the human organism is invaded by the tubercle bacillus this pathogenic phage may come into action, and, by its success or non-success in lysing the tubercle bacillus, decide whether cancer or tuberculosis shall supervene.

It is notable that tuberculosis is the enemy of the young whereas cancer attacks the middle-aged. It is also notable that the zinc content of the tissues, highest in the embryo, falls during early years and rises in later life. Is it too much to suppose that after invading tubercle bacilli have been lysed by the phages, those phages lie dormant until enlivened by the increase in the zinc content of the tissues, an increase specially notable in the nuclei controlling the multiplication of cells?

Cancer has for some time been regarded as a virus disease. The nature of that virus has hitherto been unknown. Dr. Cruickshank has been led, by logical argument based upon admitted premises, to the conclusion that there can be no explanation of the exact relation between the cancer and tuberculosis mortality rates unless the existence of a pathogenic bacteriophage, indigenous to the tubercle bacillus, be admitted. *Is that bacteriophage the virus which all cancer workers are seeking?* In other words, are Steenken's lytic factor, Cruickshank's pathogenic phage and Gye's[22] non-specific cancer factor one and the same?

That is one question, upon the answer to which may depend great issues. To that I am adding the other question which I asked a few minutes ago, about the possible relation of Meyers' cancerogenic factor with Lyle Cummins' protective factor. All through, you see, there is inescapable evidence that what favours tuberculosis opposes cancer, and vice versa; yet the rigidity with which cancer and tuberculosis continue year after year to claim, in varying proportions, 20 per cent of the total mortality forces upon one the conclusion that the relationship is close and intimate. Can there be any other explanation than that suggested by Dr. Cruickshank? And, if not, can one discuss atmospheric pollution in relation to tuberculosis without discussing at the same time its relation to cancer as well?

It may be that those who are engaged in studying the smoke problem will be able to carry the matter a stage further. Is there any factor in coal-tar which could biochemically aid the multiplication of the phages presumed by Cruickshank? Is there any other product of combustion, either solid or gaseous, which might do so? Something like 85 per cent of us are infected by tubercle bacilli, and amongst that 85 per cent there are three sub-divisions to be noted:—

1.	Those who die of tuberculosis	20%
2.	Those who die of cancer	
3.	Those who escape both	80%

Can it be shown that those in the second category have been "smoked" to a greater extent than those in the third? I think it can; I think that it has been shown already, by Bertillon, Green and others.

Remembering that coal ash contains a considerable proportion of zinc (2½%), is it possible that Kennaway's tar-contained compound "unstable and present in amounts so small as those of the vitamins in food" may operate by tipping the symbiotic balance in the direction of cancer? Professor Cook, according to a recent issue of the Daily Telegraph," stated that he and Kennaway had "arrived at a working hypothesis that "cancer may be caused by some biochemical process in the body 'taking the wrong turning' and creating some product which acts on the cells and forms cancer." Can it be that

smoke is wholly or partially responsible for upsetting the symbiotic balance and thus activating the pathogenic phages?

These, gentlemen, are all questions which need consideration if the Cruickshank hypothesis be accepted. I must emphasise that it is only a hypothesis as yet. It may be conclusively destroyed. But, if it be upheld, it must surely rank as a contribution of the utmost importance towards the final conquest of the two worst enemies of the human race —tuberculosis, the White Scourge, and cancer, the terror of the civilised world.

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Tuberculosis Control

Methods For Finding Tuberculosis In The Minimal Stage*

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ONE of the greatest defects of the present program of tuberculosis control is that in most instances the program does not begin to operate until the individual has tuberculosis in an advanced stage. Potter[1] has recently reported that although the majority of patients (64%) consulted their physician and were diagnosed tuberculous in a reasonably short time after symptoms first appeared, the disease was already in an advanced stage in 98 per cent of the cases. This condition prevails because in most instances minimal tuberculosis produces few symptoms and practically no physical signs. This fact has been demonstrated again and again. From a study on student nurses at Bellevue Hospital, New York City, Amberson[2] came to the conclusion that minimal tuberculosis can be found only by routine yearly x-ray examinations of the chest, because symptoms are not in proportion to the degree of involvement and physical signs are

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slight or lacking. Likewise in a study of 17,765 applicants for employment at the Metropolitan Life Insurance Company, Fellows[3] found that only 18 per cent of the individuals with tuberculosis could be discovered by the usual methods of examination, that is, by a careful medical history and physical examination. However, when these same applicants were examined with a fluoroscope and the persons with "abnormal chest" were given an x-ray examination four times as many cases were discovered. That is, where only 44 persons with tuberculosis were found by regular examinations, 200 additional cases were discovered by fluoroscopic and x-ray examination. It is apparent, therefore, that if tuberculosis is to be discovered in the minimal stage the physician must make use of measures of greater precision than the usual medical history and physical examination. In particular, the physician must resort to a wider use of the tuberculin test and x-ray on all patients who consult him for symptoms that even in the remotest way suggest the possibility of tuberculosis.

Although the tuberculin test and x-ray examination are given a great deal of prominence it must always be remembered that by themselves they do not afford sufficient evidence to make a diagnosis of pulmonary tuberculosis. To make a justifiable diagnosis the clinician still remains the most important factor. Even for him a reliable diagnosis must be based on all the facts secured, from the medical history, tuberculin test, x-ray physical examination and the confirmatory evidence of the clinical laboratory.

In tuberculosis as in every other disease the data obtained from a good medical history are of prime importance. A history of tuberculosis in the family should arouse suspicion although by itself it is not indicative of the presence of this disease. Tuberculosis should be particularly suspected however in all instances where there has been close personal contact with (a) a person with active tuberculosis; (b) a person with a cough of long duration or (c) a person who has died from any form of tuberculosis. Likewise, a history of repeated attacks of pneumonia, chronic bronchitis, bronchiestasis or other lung infections should be regarded with suspicion until definitely proved to be non-tuberculous. The history is especially informative in all instances where a young person has had an attack of pleurisy with effusion or haemoptysis. In fact, in such instances a provisional diagnosis of tuberculosis is amply justified, and should be discarded only after careful and repeated examinations. Any of the symptoms of tuberculosis as cough, excessive expectoration, fatigue, fever, fast pulse, indigestion, nightsweats, etc., suggest the possibility of this disease and should lead always to further examination. The age of the individual is also of considerable importance, particularly when dealing with school or other population groups. The significance of this factor has been repeatedly emphasized by Lawrason Brown who maintains that the majority of all cases of pulmonary tuberculosis can be discovered during the age period 14 to 24 years.

Of course every patient who has a remotely suggestive history can not and in fact need not be examined thoroughly for tuberculosis, because we have two very effective screens for determining which patients require future study. They are the fine screen of the tuberculin test and the course screen of the x-ray. In any examination of population groups as a whole these two screens are absolutely essential in order that efforts can be concentrated on those individuals who need attention.

The simplest and most accurate means of screening out those individuals who have acquired an infection with tubercle bacilli is the tuberculin test. A negative tuberculin test indicates (1) that the person tested has never been infected with tubercle bacilli, or (2) that a previous tuberculous lesion has completely healed and so has become obsolete. According to Opie[4], "A negative tuberculin reaction is evidence that there is no existing tuberculous infection." There are a few easily recognized exceptions to this rule. It is well known, for example, that the sensitiveness to tuberculin may be decreased or even disappear during the course of acture exanthematous diseases or during the course of other diseases associated with high temperature. Likewise a person in a moribund condition will frequently fail to react to tuberculin. The test is also negative immediately following the first infection with tubercle bacilli (during the preallergic stage) because it requires a period of from one to three weeks

for the body to become sensitized to tuberculo-protein.

A positive tuberculin test means that the person reacting has at least one focus of living tubercle bacilli in his body. This signifies that a positive reactor has acquired the primary or first infection type of tuberculosis, and it may indicate that he has the secondary or reinfection type of the disease. Unfortunately the tuberculin test does not give information regarding the age, size or type of the tuberculous lesion. An arrested primary lesion too small to be demonstrated even by x-ray, a moderate sized well-encapsulated lesion, or a large progressive lesion may produce the same degree of reaction. The tuberculin test simply does not differentiate between active and latent tuberculosis. For this reason all positive reactors particularly those between the ages 14 and 25 should be examined with x-ray to determine the type of tuberculous lesion present.

It was formerly believed that all adults and 95 per cent of the children of teen age were infected with tuberculosis. To-day these figures are much too high, the correct figures, in the United States at least, are from 50 to 75 per cent for adults and from 10 to 25 per cent for children of teen age. However, the amount of tuberculous infection is not constant throughout the country but varies considerably, even in different parts of any one state.

In Colorado examinations conducted largely among high school students during 1936 and 1937 showed an average incidence of positive reactors to be 25.8 per cent (10,796 individuals tested). In Logan County the incidence was only 8 per cent (750 tested), while in Colorado Springs it was 39.8 per cent (856 tested). In Denver the incidence of positive reactors was practically the same as for the State as a whole, that is, 26 per cent (5,400 tested). Tentative reports from our testing of approximately 1,800 students at the Colorado State College of Agriculture, at Fort Collins, show a 15 to 20 per cent incidence of positive reactors. Although these figures vary considerably they all point definitely to the fact that today much less than 100 per cent of the population react to tuberculin.

After completion of the tuberculin test all negative reactors can safely be eliminated as far as tuberculosis is concerned. The positive reactors, particularly those between the ages of 14 and 25 years, should have an x-ray film made of their lungs. The effectiveness of the x-ray has been proved many times. As the result of an extensive study made at the Trudeau Sanatorium by Brown and Sampson[5], it was concluded that as little as one cubic centimeter of tuberculous tissue will produce a visible change in density on an x-ray film of the lungs. They also showed that when the incidence of Brown's five cardinal diagnostic criteria of pulmonary tuberculosis were compared with the final diagnosis, the x-ray findings were by far the most accurate. For example when 280 consecutive minimal cases of pulmonary tuberculosis were studied it was found that 35 per cent had tubercle bacilli in the sputum, 27 per cent had rales in the upper third of one or both lungs, 26 per cent had haemoptysis and 12 per cent had pleuritic effusion. Against these, the x-ray revealed parenchymal lesions in over 99 per cent of the cases.

Furthermore, in far advanced tuberculosis the x-ray was by far the most accurate method of detecting even extensive destruction like cavity formation. Thus in 392 patients with cavities demonstrable by x-ray only 15 per cent were diagnosable by physical examination.

Although the dependability of the x-ray has been amply demonstrated it must be remembered that the x-ray film merely shows a pattern of shadow complexes which tend to assume certain characteristics in different diseases. It is apparent therefore, that accurate interpretation of these shadows requires the services of an experienced observer. It is also apparent that an accurate diagnosis of tuberculosis can not be made from the x-ray film alone—the x-ray film is merely one form of evidence leading toward the correct diagnosis. However, an x-ray film of the lungs which shows the characteristic changes of tuberculosis is such strong evidence in favor of the presence of this disease that a very careful and thorough examination should be made before the presumptive diagnosis is discarded. The most characteristic feature of the x-ray film in pulmonary tuberculosis is the occurence of parenchymal mottling in the upper third of one or both lungs. When mottling occurs in this location

it is due to tuberculosis in 95 per cent or more of all cases. This does not mean that tuberculosis can not produce parenchymal mottling in other portions of the lungs, it simply means that when parenchymal mottling occurs in the upper third of the lungs it is most likely due to tuberculosis. For accurate diagnostic work, the stero-film is superior to the single film, particularly since in the single film small areas of disease are frequently obscured by rib shadows. However, Myers[6] maintains, and I believe rightly, that a single film of the chest, when properly made and carefully interpreted, rarely misses significant tuberculous pathology. This is particularly true when serial films are taken over a reasonable period of time (6 months to 1 year).

Although physical examination is of very limited value in dealing with minimal pulmonary tuberculosis, this procedure should not be neglected. Experience has shown that the most reliable physical findings in pulmonary tuberculosis are rales located in the upper third of one or both lungs. Whenever moderately coarse rales in the upper third of the lung persist after cough they indicate tuberculosis in over 90 per cent of all cases. Thus in a study of 1,000 patients Brown found that 99.3 per cent of those having rales in this area also showed parenchymal lesions by x-ray, which were proved to be tuberculous. It is apparent, therefore, that persistent rales in the upper third of the chest should always suggest pulmonary tuberculosis.

From a public health, as well as a diagnostic standpoint, the most important evidence of tuberculosis is the demonstration of tubercle bacilli in the sputum. According to Plunkett[7], "From a point of diagnosis, the demonstration of tubercle bacilli in the sputum is the only method of unequivocal value. All other tests or examinations belong in the field of presumptive evidence." Likewise from the public health viewpoint the examination of the sputum is most important because the sputum is the vehicle for the transmission of this disease, and so shows which patients require isolation. Examination of the sputum is also important in determining the proper course of treatment for a patient, as well as indicating the prognosis. Thus if the sputum contains large numbers of bacilli it is very suggestive of cavity formation and extension of the disease. Also thin watery sputum which contains many tubercle bacilli is extremely dangerous since it promotes extension of the disease by bronchogenic dissemination. Likewise the more bacilli present and the longer they remain present, the worse the prognosis. In other words, adequate sputum examination is one of the most important steps in arriving at a correct diagnosis in making a reasonable prognosis in determining the proper course of treatment and in protecting the health of any community. So important is this examination that most States, including Colorado, have established efficient laboratories where such examinations can be secured without cost to the physician. "Nevertheless," says Plunkett, "it is astounding that of 65,335 reports of pulmonary tuberculosis received by the New York State Department of Health in the past nine years, less than one-half (about 45%) of the cards showed that the patient had had a sputum examination." Unfortunately the same condition prevails in Colorado. For example, in going over the death reports from Weld County, I was surprised to find that out of the 41 deaths ascribed to tuberculosis in 1935-1936, not a single diagnosis had been confirmed by examination of the sputum. Although the sputum is frequently negative in minimal tuberculosis it was shown to be positive in 35 per cent of the minimal cases examined at Trudeau Sanatorium. With such a high percentage of positive sputum in even minimal tuberculosis the great importance of sputum examination is evident. In connection with sputum examination for tubercle bacilli it should always be remembered that no sputum can be said to be truly negative unless it has successfully run the gauntlet of microscope and culture or animal inoculation.

In summary then, the chief means of making a diagnosis of pulmonary tuberculosis, particularly in the minimal stage are: 1. A good medical history. 2. A tuberculin test. 3. An x-ray film of the chest. 4. A physical examination. 5. Laboratory examinations especially of the sputum and, 6. The services of a thoughtful clinician to interpret the findings secured by all these means.

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Lung Abscess*

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THE first case we have today is a lady 26 years of age, school teacher by profession, was first seen by me on September 12, 1936, complaining of pain in upper right chest, cough, expectorating a foul smelling sputum, chills and fever. The patient stated that after returning from a long automobile trip she had her tonsils removed under general anesthesia (lowered resistance plus infection responsible for many ills). Five days later she began to have severe pain in her right shoulder and around her right breast. This lasted for three days, two days later she began to have chills and fever.

She was first seen by me, two weeks after the operation. Signs suggested abscess of upper lobe of right lung. She was ordered in the hospital. Laboratory report showed a hemoglobin of 60 per cent, erythrocytes 3,280,000, leukocytes 15,300, polynuclears 80 per cent, small lymphocytes 18 per cent, large lymphocytes 1 per cent, basophiles 1 per cent. Wassermann negative, sputum positive for Vincents organism. X-ray picture made on Sept. 13th revealed a shadow in upper lobe of right lung, consistent with a suppurative process. Artificial pneumothorax was advised and was immediately started. On Sept. 16, her hemoglobin dropped to 55 per cent. She was given 400 cc. of citrated blood on the 18th, again on the 21st and 26th. During this time she received four air injections. She ran a stormy time for about ten days. The abscess healed by resolution and she was discharged from the hospital on Oct. 2, 1936.

Two or three days before leaving the hospital, patient began to complain of pain in lower left chest and left shoulder. A few squeaking rales were heard over lower left lobe but x-ray picture made at this time revealed no pathology. The pain in left chest continued. About a week after leaving the hospital she developed fever and cough. A few days later she coughed up some bloody mucous. She re-entered the hospital Oct. 16th complaining of pain in lower left chest, cough, expectoration and fever. Sputum negative for Vincents organism. An x-ray picture made on this date showed a density in lower left lobe suggestive of a pneumonitis. I considered collapsing this lung but my confrere advised me to wait and give her a chance. On Oct. 21st, five days later, another picture was made which showed an area organized and undergoing suppuration. Pneumothorax was started at once on the left side. She got along nicely and was doing so well she wished to go home. She was discharged on Nov. 9th with a diagnosis of abscess of lower left lobe being well collapsed by artificial pneumothorax. On Nov. 12, about 5 A. M., during a coughing spell, patient was taken with a severe pain in lower left chest, stabbing in character. I was called to see her about 12 hours later. Patient was in severe pain, very rapid pulse, temperature 102, distended abdomen and very tender in region of diaphragm. I thought she had a diaphragmatic pleurisy, gave 200 cc. of air and left her. She had a good night but was taken worse the next day and brought back to the hospital on Nov. 13th about 8 P. M. in profused shock, very short of breath, cyanosed, rapid thready pulse, cold clammy sweat, left chest tympanitic with dullness at base, thoracic viscera pushed to the right. A needle was inserted and 350 cc. of cloudy foul smelling fluid was aspirated. What had

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happened, during a coughing spell she had ruptured her parietal pleura, probably the size of a pin point, but enough to infect her pleural cavity. Spontaneous pneumothorax and empyema was the result. Two days later she was aspirated again and transfused. The following day, Dr. Payne put in a drain, under local anesthesia. The patient had a stormy but complete recovery. Discharged from the hospital Feb. 28, 1937 as cured.

I think I made two mistakes in this case. First, as soon as the inflammatory area was recognized in the left lung it should have been collapsed. Second, I let her go home too soon. Absolute rest in bed is essential until they are symptom free.

Case number two is a white, male, age 42, produce merchant, was first seen Oct. 9, 1936, complaining of cough, expectorating a large quantity of foul smelling substance, very weak and drowsy and a rapid loss of weight.

The patient stated that he developed an abscessed tooth the last of June 1936, was treated by a dentist. During the course of treatment he developed a cellulitis of jaw and neck which had to be opened and drained. After swelling had subsided, the tooth was extracted. About two weeks after the extraction of the tooth he developed a cough and began to raise large quantities of foul smelling sputum which has continued. A diagnosis of abscess of lower lobe of right lung was made and advised hospitalization for pneumothorax treatment. He entered the hospital Oct. 16th 1936 with a hemoglobin of 67 per cent, red cells 4,300,000, white cells 12,200, polynuclears 76 per cent, small lymphocytes 22 per cent, sputum negative for tubercle bacilli and Vincents organism. He was given a transfusion and pneumothorax started on the right side Oct. 17th. He was discharged from the hospital Oct. 30th doing nicely with the lower lobe of right lung, which was the diseased area, well collapsed. The treatment was continued at home once a week. About five weeks later he was requested to go to the hospital for an x-ray of chest. While on his way to the hospital he had a severe coughing spell followed by a severe pain in right chest. He was taken so sick he had to return home. He re-entered the hospital on Dec. 10, 1936 with a spontaneous pneumothorax and empyema. Dr. George Schench put in an open drainage on Dec. 15th. Following this he had a stormy time but made a complete recovery, discharged Jan. 25, 1937. I made a mistake in this case by letting him leave his bed once he was at home.

I have been treating acute abscess of the lung with pneumothorax since 1920—have treated close to 40 cases. These are the only two cases that ruptured in the pleural cavity.

Case No. 4, Mrs. D. B., age 25, waitress, complaint: general weakness, expectorating bloody mucous. Consulted me Feb. 8, 1937. Patient stated that around middle of December she was taken with Grippe. Had fever for about 4 days with a non-productive cough. About 10 days ago she began to expectorate bloody mucous with a foul odor. About a week ago she coughed up about a pint of foul smelling pus. Examination revealed an abscess of right lung. She was referred to the Norfolk General Clinic and placed in Dr. Hodges' charge. X-ray of chest revealed an abscess in the hilus region of the right lung, apparently draining into a bronchus. She was put to bed on a high protein diet and cleared up in 3 weeks. Discharged as cured.

Incidence

Reviewing the records in St. Vincents Hospital for the past 10 years we find 56 cases of lung abscess. For the same period of time in Norfolk General Hospital we find 36 cases. Of the first group of 56 cases there were 27 deaths or 48.2 per cent. Of this group eleven were given the benefit of some form of surgery with two cured, three deaths, two discharged unimproved, and four improved. Thirty nine were treated with rest and symptomatically, with 19 deaths, 15 being discharged as improved and 5 cured. Five were treated with collapse therapy, 4 died and 1 improved.

Of the second group, 3 were operated on with 2 deaths and 1 cured. Twenty were treated medically, 4 died, 3 cured, 7 were discharged as improved and 6 unimproved. Thirteen were treated with artificial pneumothorax, there were 3 deaths, 3 cured, 5 being discharged as improved with treatments continued on the outside, 2 as unimproved.

Etiology

It may be aspiratory, foreign body or septic

NORFOLK GENERAL HOSPITAL

CASE NUMBER	SEX	RACE	AGE	CAUSE	TREATMENT	DURATION IN HOSPITAL	TRANSFUSION	RESULT	NEOSALVERSAN FOR POSITIVE VINCENT'S
					1926				
3084 (2)	Male	White	16 Yrs.	Probably T. B.	Rest in bed	42 days	No	Improved	Neg.
1085	Female	White	23 Yrs.	Syphilis, Tertiary	Rest in bed	2 days	No	Improved	Neg.
3865	Female	Colored	31 Yrs.	Grippe	Rest in bed	10 days	No	Cured	Neg.
4190	Male	White	36 Yrs.	Pneumonia & T. B.	Rest in bed	3 days	No	Unimproved	Neg.
2395	Male	White	58 Yrs.	Gan. Appendicitis	Rest in bed	5 days	No	Unimproved	Neg.
					1927 None				
					1928				
4114	Female	White	13 Yrs.	Tonsillectomy	Rest in bed	25 days	No	Improved	Neg.
2513 (2)	Female	White	65 Yrs.	Sinus Operation	Pneumothorax	19 days	No	Improved	Neg.
3748	Male	White	23 Yrs.	Pneumonia	Resection, 9th rib	18 days	No	*Death*	Neg.
3094	Male	White	39 Yrs.	Bronchopneumonia	Rest in bed	6 days	No	*Death*	Neg.
1630	Male	White	3 Yrs.	Tonsillectomy	Observation	3 days	No	Unimproved	Neg.
					1929				
4341	Male	White	58 Yrs.	Sinus Operation	Rest in bed	9 days	No	Improved	Neg.
					1930				
3737	Female	White	76 Yrs.	Bronchopneumonia	Rest in bed	11 days	No	*Death*	Neg.
					1931				
3362	Female	White	59 Yrs.	Pneumonia	Rest in bed	10 days	No	Cured	Neg.
					1932				
5018	Male	White	15 Yrs.	Sinus Operation	Rest in bed	27 days	Yes	*Death*	Neg.
1174	Female	White	49 Yrs.	Probably T. B.	Rest in bed	7 days	No	Unimproved	Neg.
5796	Female	White	29 Yrs.	Miscarriage	Pneumothorax	22 days	No	Improved	Pos.
2196	Male	White	47 Yrs.	Bronchopneumonia	Resection, 6th rib	17 days	No	*Death*	Neg.
					1933				
356	Male	White	59 Yrs.	Extraction, Teeth	Collapse	59 days	No	*Death*	Pos.
662	Male	White	37 Yrs.	Pneumonia	Rest in bed	8 days	No	Improved	Neg.
12200	Male	White	48 Yrs.	Grippe	Rest in bed	3 days	No	Unimproved	Neg.
12172	Male	White	51 Yrs.	Gastric Ulcer	Pneumothorax	2 days	No	Unimproved	Neg.
					1934				
15320	Male	White	24 Yrs.	Tonsillectomy	Pneumothorax	44 days	No	*Death*	Pos.
17083 (3)	Male	White	17 Yrs.	Probably T. B.	Pneumothorax	19 days	No	Cured	Neg.
15585 (2)	Male	Colored	23 Yrs.	Appendicitis, Acute	Rest in bed	5 days	No	Unimproved	Neg.
21980 (7)	Male	White	18 Yrs.	Probably T. B.	Pneumothorax	145 days	No	*Death*	Pos.
					1935				
21187	Female	White	33 Yrs.	Miscarriage	Rest in bed	14 days	Yes	Improved	Neg.
19444	Male	White	39 Yrs.	Oral Sepsis	Pneumothorax	87 days	No	Cured	Pos.
20372	Male	White	29 Yrs.	Pneumonia	Pneumothorax	58 days	No	Improved	Pos.
21786	Female	White	24 Yrs.	Tuberculosis	Pneumothorax	7 days	No	Unimproved	Neg.
					1936				
28534	Female	White	25 Yrs.	Tonsillectomy	Pneumothorax	128 days	Yes	Cured	Neg.
28058	Female	White	25 Yrs.	Tonsillectomy	Pneumothorax	23 days	No	Improved	Neg.
23983	Male	White	16 Yrs.	Pneumonia	Res., 8 or 9th ribs	49 days	No	Cured	Neg.
25222	Female	White	30 Yrs.	Secondary Anemia	Rest in bed	2 days	No	Improved	Neg.
28053	Male	White	42 Yrs.	Abscess Tooth	Pneumothorax	14 days	Yes	Improved	Neg.
27517	Female	White	16 Yrs.	Tonsillectomy	Rest in bed	22 days	No	Cured	Neg.
23806	Male	Colored	52 Yrs.	Pneumonia	Rest in bed	43 days	Yes	*Death*	Neg.

ST VINCENT'S HOSPITAL

					1926				
753	Male	Colored	34 Yrs.	Stricture Esophagus	Rest in bed	16 days	No	*Death*	Neg.
4437	Male	Colored	13 Yrs.	Bronchopneumonia	Rest in bed	31 days	No	Improved	Neg.
					1927				
5522	Male	White	40 Yrs.	Lobar Pneumonia	Rest in bed	41 days	No	*Death*	Neg.
5800	Male	White	32 Yrs.	Unresolved Pneumonia	Pneumothorax	11 days	No	Improved	Neg.
6168	Male	White	17 Yrs.	Bronchitis, Acute	Rest in bed	26 days	No	Cured	Neg.
8391	Male	White	50 Yrs.	Appendicitis, Gan.	Rest in bed	36 days	Yes	*Death*	Neg.
8525	Female	White	22 Yrs.	Bronchopneumonia	Rest in bed	2 days	No	*Death*	Neg.
8527	Male	White	30 Yrs.	Lacerations, Face & Neck	Rest in bed	11 days	No	*Death*	Neg.

CASE NUMBER	SEX	RACE	AGE	CAUSE	TREATMENT	DURATION IN HOSPITAL	TRANSFUSION	RESULT	NEOSALVERSAN FOR POSITIVE VINCENT'S
8808	Female	Colored	47 Yrs.	Syphilis	Rest in bed	30 days	No	Improved	Neg.
8983	Female	White	23 Yrs.	Tonsillectomy	Rest in bed	20 days	No	Improved	Neg.
					1928				
9999	Female	White	49 Yrs.	Laparotomy	Aspiration	10 days	No	Improved	Neg.
10466	Male	White	30 Yrs.	Pneumonia	Diagnostic Punch	21 days	No	Improved	Neg.
12659	Female	White	12 Yrs.	Lobar Pneumonia	Rest in bed	36 days	No	Improved	Neg.
12556	Female	Colored	18 Mos.	Bronchopneumonia	Incision & Drainage	8 days	No	Unimproved	Neg.
					1929				
14471	Female	White	13 Yrs.	Mastoidectomy	Rest in bed	10 days	Yes	*Death*	Neg.
16197	Female	White	29 Yrs.	Lobar Pneumonia	Rest in bed	2 days	No	*Death*	Neg.
17460	Female	White	23 Yrs.	Tonsillectomy	Rest in bed	8 days	No	Cured	Neg.
18008	Male	White	20 Yrs.	Drainage, Abscess, Liver	Rest in bed	19 days	No	Improved	Neg.
					1930				
18373	Female	White	28 Yrs.	Pregnancy, O. D. A.	Incision & Drainage	18 days	No	*Death*	Pos.?
19563	Male	White	36 Yrs.	Pneumonia	Resection, 8th rib	14 days	No	Cured	Neg.
19365	Male	Colored	43 Yrs.	Frac. Bones of Face	Rest in bed	7 days	No	*Death*	Neg.
19626	Male	Colored	32 Yrs.	Lobar Pneumonia	Resection, 9th rib	26 days	No	Cured	Neg.
20222	Male	White	36 Yrs.	Septicemia, Gen.	Aspiration	6 days	No	*Death*	Neg.
20392	Female	White	19 Yrs.	Appendicitis, Rup.	Resection, 8th rib	15 days	No	*Death*	Neg.
20872 (2)	Female	White	19 Yrs.	Pneumonia	Pneumothorax	47 days	No	*Death*	Neg.
20603	Male	Colored	49 Yrs.	Syphilis, Tertiary	Pneumothorax	7 days	No	*Death*	Neg.
21867	Male	White	28 Yrs.	Pneumonia	Resection, rib	20 days	No	Improved	Neg.
					1931				
23574	Male	White	18 Yrs.	Lobar Pneumonia	Resection, 5th rib	78 days	No	Improved	Neg.
24525	Female	White	40 Yrs.	Pneumonia	Rest in bed	8 days	No	*Death*	Neg.
28084	Male	Colored	39 Yrs.	Pneumonia	Pneumothorax	66 days	No	*Death*	Pos.?
25927	Male	White	48 Yrs.	Empyema, Chronic	Rest in bed	23 days	No	*Death*	Neg.
					1932				
28054	Male	White	28 Yrs.	Pleurisy	Rest in bed	11 days	No	Cured	Neg.
28043	Male	White	10 Yrs.	Foreign Body, Bronchus	Rest in bed	11 days	No	*Death*	Neg.
28460	Male	White	30 Yrs.	Com. Frac. Femur	Aspiration	7 days	No	*Death*	Neg.
28796	Female	Colored	43 Yrs.	Osteomyelitis, Jaw	Postural Drainage	9 days	No	*Death*	Pos.
27208	Female	White	49 Yrs.	Stroke Paralysis	Rest in bed	31 days	No	Improved	Neg.
26881	Male	Colored	26 Yrs.	Bronchopneumonia	Rest in bed	13 days	No	*Death*	Neg.
					1933				
29981	Female	White	21 Yrs.	Bronchopneumonia	Postural Drainage	13 days	No	Improved	Neg.
					1934				
34645	Male	White	24 Yrs.	Tonsillectomy	Rest in bed	19 days	No	Improved	Neg.
34940	Female	White	28 Yrs.	Lobar Pneumonia	Rest in bed	36 days	No	Cured	Neg.
35867	Male	Colored	45 Yrs.	Fracture Patella	Rest in bed	2 days	No	*Death*	Neg.
					1935				
32129	Male	White	40 Yrs.	Conculsions	Rest in bed	5 days	No	Improved	Neg.
31657	Male	Colored	25 Yrs.	Gunshot Wound, Chest	Rest in bed	31 days	Yes	*Death*	Neg.
36312	Male	Colored	49 Yrs.	Advanced T. B.	Rest in bed	92 days	No	Improved	Neg.
37481	Male	White	22 Mos.	Bronchopneumonia	Rest in bed	5 days	No	Improved	Neg.
38241	Male	Colored	49 Yrs.	Fuso-Spirochital	Postural Drainage	7 days	No	Improved	Neg.
37940	Male	Colored	65 Yrs.	Far Advanced T. B.	Rest in bed	23 days	No	Improved	Neg.
38686	Male	Colored	60 Yrs.	Bronchopneumonia	Pneumothorax	10 days	No	*Death*	Neg.
39724	Male	Colored	32 Yrs.	Pneumonia	Rest in bed	12 days	No	Improved	Neg.
40751	Male	White	11 Yrs.	Gunshot Wound, Leg	Rest in bed	11 days	No	Cured	Neg.
					1936				
40579	Male	Colored	54 Yrs.	Pyorrhea	Rest in bed	33 days	Yes	*Death*	Pos.
41252	Male	White	48 Yrs.	Pulmonary T. B.	Rest in bed	4 days	No	*Death*	Neg.
41957	Female	White	25 Yrs.	Pento-baritol Poisoning	Rest in bed	48 days	Yes	*Death*	Neg.
42969	Male	Colored	40 Yrs.	Pneumonia	Incision & drainage	8 days	No	Improved	Neg.
43519	Male	Colored	80 Yrs.	Swallowed Piece wood	Rest in bed	11 days	No	*Death*	Neg.
43124	Male	Colored	35 Yrs.	Probably T. B.	Rest in bed	5 days	No	Improved	Neg.

material from mouth or pharynx, embolic or unresolved pneumonia.

Treatment

Rest in bed is essential until symptom free, using postural drainage when possible. Transfuse if hemoglobin is below 70 per cent. Neosalvarsan if Vincents organisms are found in the sputum. Broncoscopy is advisable in order to rule out foreign body and if the abscess be located near a bronchus better drainage would be promoted.

Collapse therapy has given the best results in my hands. In order for pneumothorax treatment to be successful it should be started before adhesions have had a chance to form.

About fifty per cent of cases of lung abscess will heal by rest in bed and symptomatic treatment especially if located in upper lobes. The remaining 50 per cent will either die or advance into a chronic state. So I advise collapsing early to save as many of the remaining 50 per cent as possible.

If you are unable to collapse the lung on account of adhesions then one must resort to surgery, put in open drainage, but surgery should not be attempted until the abscess is walled off and the lung is well anchored to the chest wall.

Pulmonary Tuberculosis – Who Has It!

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FOR many years institutions for the care of pulmonary tuberculosis have been reporting on the number of early or minimal cases admitted for treatment and many papers have been presented stressing the importance of early diagnosis. Yet, today, the percentage of such minimal cases in sanatoria is about equal to that experienced by the first state sanatorium ever established—Massachusetts State Sanatorium, Rutland, Massachusetts. According to Whitney[1] the early cases admitted to 274 civilian sanatoria covering the year 1931 were 16 per cent of the total admitted.

Everyone agrees that the minimal case has almost one hundred per cent chances for complete recovery if promptly informed of the condition and if given competent medical advice which can be completely carried out and over a sufficient period of time. Tuberculosis societies and organizations have for years taught the public the value of early diagnosis and stressed symptoms which should cause persons to suspect the presence of early tuberculosis. Still no improvement worthy of note has resulted.

When does pulmonary tuberculosis develop, so that an early diagnosis can be made? According to Amberson[2] eighty-three per cent of one hundred and forty-two previously healthy persons developed tuberculosis between the ages of eighteen and twenty-seven years. According to Hetherington[3] about one per cent of tuberculin positive boys of high school age have apical infiltrations demonstrable by x-ray and about two per cent of girls of similar ages.

When does pulmonary tuberculosis produce symptoms? This is an important question since at present most persons apparently believe there are symptoms produced by early tuberculosis and most of the educational literature tends at least to back up this impression by posters and slogans such as "Suspect early tuberculosis if tired, losing weight, running afternoon temperature, having persistant cough or spitting blood," etc. Personally, we believe that in most instances early tuberculosis develops and passes through to the advanced stages before such symptoms develop to the point where they serve as warning signs of trouble. In fact, we question whether early diagnosis is going to follow in increasing degree if young adults in their late 'teen ages and early twenties are advised to wait for suspicious symptoms of trouble before examination.

This opinion seems to be amply justified by Amberson[2] when he states "the early lesion will be discovered in only a small minority of cases unless the disease is viewed as a community problem and organized searches for it are made periodically. The patient, having few or no symptoms of illness, does not seek the physician." And again, Hetherington[3] states "The importance of the diagnosis

of pulmonary tuberculosis through history, symptoms and physicial signs cannot be overestimated, but unfortunately symptoms sufficiently severe to cause concern often lag far behind the anatomic involvement, so that the majority of patients present moderately advanced or far advanced disease when they first seek medical attention."

It would seem, therefore, that early diagnosis requires the examination of large numbers of apparently healthy persons of the age group living at that time of life when early lesions begin—18 to 25 or 27 years of age.

What kind of examination? We still see the physician who advises that, since his physical examination fails to disclose abnormalities an x-ray of the chest is useless. Perhaps this attitude is less than formerly—I sincerely hope so. Certainly the era of the old-fashioned clinic day is passed when twenty or more babies, children, and adults were inspected, percussed and auscultated and advised.

I, personally, had the misfortune to diagnose early pulmonary tuberculosis, twenty years ago, in the case of a physician's wife based on fatigue, afternoon rise in temperature and moist rales over an apex; diagnosis confirmed same day by an older chest expert; only to have the rash of measles the next day make the corrected diagnosis. Fortunately no great damage was done by the mistake excepting to my pride, but it did teach something.

I know of a twenty year old girl who was treated for pulmonary tuberculosis for five years because of an all day tuberculosis clinic diagnosis based on rise of temperature to 99.8° and a few obscure rales in one apex. The corrected diagnosis was made following a negative x-ray film and removal of tonsils but after five years of wasted time and effort to say nothing of wasted money.

These two instances record the dangers of making a positive diagnosis based upon history, symptoms and physical examination without confirmatory x-ray films. But the failures are more often made by overlooking early lesions in young apparently healthy individuals because an x-ray is not taken.

Hetherington[3] states "X-ray examination is necessary to determine whether or not the infection has resulted in a significant lesion." Amberson[2] states "tuberculin testing and x-ray examination of the chest, wisely planned and applied, are *indispensable* parts of the diagnostic method."

Who then should have an x-ray examination? We can all agree that all those persons now in intimate association with a known case of pulmonary tuberculosis should be most carefully and repeatedly examined since more tuberculosis will develop among these contacts than in any other group. The point of importance to be stressed here is that this group should be repeatedly examined by x-ray for many years after exposure to infection. One examination is not enough. It only states the condition at the time and does not take into account future possibilities.

While the examination by x-ray of all contacts is of most importance, that in itself is not sufficient. There are many infected young people not known to be contacts and they represent a large group among whom many cases of early pulmonary tuberculosis will occur. As stated above by Hetherington, one per cent among boys and two per cent among girls may be expected to have lesions demonstrated by x-ray provided they are known tuberculin reactors.

It, therefore, is necessary to determine which, if not all of these boys and girls, need x-ray films. Rarely are conditions such as will finance the taking of x-ray films of all students needing investigation. However, a simple and inexpensive method is available to screen out those not requiring x-ray films and that is, of course, the tuberculin skin test.

Discussion

The practising physician under the usual circumstances should not be expected to make a diagnosis of early pulmonary tuberculosis on persons coming for examination complaining of symptoms. Past criticism directed to him is not justified, since the disease usually has already passed to the advanced or moderately advanced stage. Physicians should not expect to detect early lesions by any physical examination, since it is agreed that no one can detect these lesions without the help of the x-ray.

Tuberculin testing of large numbers of contacts and of large numbers of apparently healthy individuals at the age when early

tuberculosis is known to have its beginning will result in determining those who should have x-ray examinations now and who should have them repeated annually until about thirty years of age.

This routine annual x-ray check-up should disclose those with beginning pulmonary tuberculosis when nearly all can be restored to good health without serious and prolonged treatment.

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2. *Amberson, J. Burns:* "The Lasting Cure of Early Pulmonary Tuberculosis," Journal A.M.A. Vol. 109, page 1941—Dec. 11, 1937.

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Detoxication of Tissues in Active Pulmonary Tuberculosis

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IN our battle against the toxins eliminated from an active pulmonary tuberculous focus and distributed through the various transportation systems to the tissues of the body, we are compelled to fight on three main fronts, viz., (1) to eliminate the focus itself, (2) to minimize the accessibility of the toxins to the circulation, and (3) to alter the body tissues in such a manner as to render them the least vulnerable to the toxins which had succeeded in eluding the first two lines of defense. The success of this strategy depends upon the perfect co-ordination of all the three fronts as their activities are interdependent. In the absence of any biochemical agent that would rank as a specific we are compelled to fall back on the defensive forces of the tissues and mobilize and utilize them to the highest degree for our purpose.

Of all the procedures which were designed to accomplish this end, none can be compaired for dramatic effectiveness with the application of collapse therapy. It is by no means self-evident as to just how this form of therapy accomplishes, in such a striking manner, the amelioration of symptoms, and even clinical cures of the lesion. Various theories have been advanced to interpret this phenomenon, but none of them proved satisfactory. They are delphic in their ambiguity. They explain everything and nothing.

Without dwelling longer on this theoretical, if highly important theme, I wish to mention two views that seem to stand out prominently for their clarity, (1) that the prime effect of pulmonary collapse is reduction in the amount of toxin given over from the diseased lung to the general circulation, and, (2) that the chief agency in the dissemination of toxins is the respiratory movement of the lung; that by means of its excursions the lung acts as a veritable pump for the distribution of its liquid contents; that, given an intra-pulmonary reservoir of poison open to the general circulation, its contents will be mobilized at a rate proportionate to the amplitude and rapidity of respiratory movement. If the propositions announced above be admitted, it follows that the distribution of toxins from a diseased focus will be brought to its minimum by inhibiting motion of the part. Collapse therapy accomplishes both—it closes the reservoir, and checks the pumping of the tissues into the circulation.

H. Von Hayek[1] points out that, "every method of treatment of the local organic process can be of certain value only if healing of the main focus is achieved without producing unfavorable effect elsewhere." Indeed, collapse therapy alone, without the aid of the defensive forces of the home front could hardly be successful. Of all the measures at our command to maintain the defensive forces of the body at their height of efficiency, rest (physical, mental, and emotional) is by far the most important. The more effectively the body is immobilized, the less is the amplitude and the rate of the respiratory excursion, the slower and the more even is the circulation of the body fluids, and, hence, there is less pumping of and distribution of toxins. Furthermore, the immobilization minimizes the expenditure of body

energy associated with motion or effort, thus preventing the waste of elements which must involve at least some of the factors in the cure of tuberculosis.

Conventional bed rest alone is often insufficient, as is demonstrated by the statistics of the Barnes Hospital where, from 1918 to 1927, 90 per cent of the 1,454 patients receiving it were dead in five years. In a previous article[2] I reported results with the Paterson complete immobilization treatment, where a patient is kept under a regime more rigid than one used in the cure of severe typhoid fever cases. The treatment was applied only in far advanced cases, and only after all other means suitable for those cases had been tried previously without benefit. The results obtained proved quite convincingly the great superiority of the treatment over the conventional bed rest.

It is not within the scope of this article to discuss the various methods of collapse therapy as to their respective advantages and disadvantages, indications and contra-indications, and complications. Suffice it to say that while not so many years ago, the procedure was applicable to only two per cent of the cases, now, with the marvelous improvements of technique of thoracic surgery and pneumoperitonium, there are comparatively few cases that could not be benefited by collapse therapy when judiciously applied and especially when combined with adequate bed rest.

An abundance of statistical data is compelling the conclusion on the part of clinicians that the collapse therapy should be applied early. A few weeks before excavation and bronchiogenic spread occur in the early lesion, is the golden opportunity for instituting intensive and effective treatment.

As to the rather voluminous group of biochemical agents comprising various tuberculins, vaccines, calcium, gold, heliotherapy, radiotherapy, and hormones, this group enjoyed a highly colorful and romantic history. The lure to penetrate the thick jungle which harbors the immunological mysteries of tuberculosis has captivated numerous enthusiasts. The net results obtained from the application of biochemical agents have so far proved to be rather meager when compared with those of the collapse therapy. At best, they are indicated only in individual cases, and then only as adjuvants.

Few problems have received as much attention and have been the subject of so much controversy as the matter of climatology in tuberculosis.

The medical reports of the various sanatoria situated in widely diversified climates where collapse therapy is being properly applied, seem to differ very little as to end results. Apparently climatic factors are important only in so far as they satisfy the financial, social and cultural requirements of the patient, thus providing him with a maximum of mental rest and contentment.

Almost the same comment can be made with regard to dietetics. J. Burns Amberson Jr.[3], proposes the following criteria as the measure of success in treatment, viz., "the return of the patient to normal living and normal conditions in a minimum of time. Treatment in tuberculosis is considered good if it achieves local healing, abates toxemia and permits return of normal function. But such results fail under the test of time. Results, good or otherwise, can not be evaluated short of five years; ten years are better. Superalimentation used to be thought good, except that in five years most of the patients, who had bloomed out, chiefly on this alone, were dead." Any diet that is nutritious and adjusted to the digestive capacity of the patient, without overtaxing it, is good.

In conclusion I wish to say that the aim of this article is to point out the rationale of the collapse therapy, with the hope of weakening the skepticism towards thoracic surgery, which unfortunately is still the attitude of many physicians; that in order to detoxicate the body tissues we should think more in terms of cubic centimeters of air in the pleural space than of food calories, more of rest hours than of tuberculin dilutions, and by all means, to realize that essentially thoracic surgery is a splint applied to a damaged lung, and that the urgency of applying it EARLY is just as great as in a case of a fractured limb.

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Tuberculin Tests in State 4-H Club Health Contestants*

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AT THE annual State 4-H Roundups in 1936 and 1937 the health contestants were examined by the Student Health Service, Kansas State College. These health contestants were farm boys and girls of high school age selected through physical examinations in their respective counties to compete in the state health contest. Each county is limited to one male and one female health entry.

As a part of the comprehensive physical examination tuberculin tests were made on each contestant. Before 1936 tuberculin tests were not included as a part of the state health contest. Through the cooperation of Mr. M. H. Coe, state 4-H Club leader, Kansas became the first state, as far as we can ascertain, to introduce routine tuberculin testing of 4-H state health contestants followed by chest x-rays of all positive reactors.

In 1936 the tuberculin tests made by the intradermal injection of 0.1 milligram of old tuberculin. In 1937 the tests were made by the intradermal injection of 0.0005 milligram of purified protein derivative. This amount of purified protein derivative corresponds to the amount of old tuberculin used in the previous examination and has been recommended by Hall[1] and referred to by him as the intermediate dilution of purified protein derivative.

Each year the results of the tests were read 48 hours after the injections were made. The results were classified according to the following method: *Negative*—absence of redness or swelling at the site of injection. *1 plus*—the appearance of an area of swelling between 0.5 and 1.0 centimeter in its greatest diameter. *2 plus*— the appearance of an area of swelling with its greatest diameter between 1.0 and 2.0 centimeters. *3 plus*—the appearance of an area of swelling with its greatest diameter more than 2.0 centimeters. *4 plus*—the appearance of an area of swelling with definite necrosis. This classification is modified from the one given by the National Tuberculosis Association[2].

In 1936 there were 141 contestants with 13 or 9.2 positive reactors. In 1937 there were 117 contestants with 21 or 17.9 per cent positive reactors. Probably the higher percentage of positive reactors found in 1937 is due to the use of a better standardized preparation of tuberculoprotein. Each year there was only one undesirable reaction (4 plus) in the group tested.

The homes of the positive reactors of these groups of boys and girls are fairly well distributed throughout the state.

Each year chest x-rays were made of each positive reactor. We are greatly indebted to Dr. Galen M. Tice, radiologist at the University of Kansas Medical School, for the interpretation of the x-ray plates. Of the 13 cases x-rayed in 1936 there were 10 that showed no roentgenological evidence of tuberculous lung infection, 2 that showed arrested childhood type of tuberculous lung infection and 1 that showed old pathological lung changes of non-specific etiology. Of the 21 cases x-rayed in 1937 there were 9 that showed no roentgenological evidence of tuberculous lung infection, 10 that showed arrested childhood type of tuberculous lung infection, and one that showed old pathological lung changes of non-specific etiology. There were no active cases of the childhood type of tuberculosis. No cases of the adult type of tuberculous infection were encountered; but it should be emphasized that during the next decade the individuals in this group of tuberculous infections are much more likely to develop tuberculosis than would the individuals of a similar non-infected group, as pointed out by Myers and Tarrington[3]. For this reason we made a uniform deduction in the health score of each positive reactor. In carrying out this procedure we were aware, of course, that this view is not uniformly held by workers in this field[4].

Each year the x-ray reports of each contestant were sent to their parents by the 4-H state office. This action is in accordance with

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** From the Student Health Service, Kansas State College.

the main purpose of these special examinations, namely, the dissemination of public health education in modern methods of diagnosis and control of tuberculosis to an intelligent and influential section of the rural population. It is hoped that this tuberculosis program will be adopted by other state 4-H Clubs.

Summary

1. Tuberculin testing with chest x-rays of all positive reactors has been introduced to an important group of the Kansas farm population.

2. Superior general health and absence of physical defects apparently do not appreciably diminish the incidence of tuberculous infection.

3. The one-test method with the intermediate dilution of purified protein derivative apparently detects cases of tuberculous infection with a high degree of accuracy.

4. In 1936 and 1937, deductions have been made in the health scores of 4-H Club state health contestants who had positive tuberculin reactions. It may be found feasible to extend generally this policy of deduction for positive tuberculin reactors to health and insurance examinations.

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Organization News

Named Superintendent

Dr. Earl E. Carpenter, Superior, Wisconsin, a Fellow of the American College of Chest Physicians, has been named superintendent of the Middle River Sanatorium on Feb. 4th.

Division of Tuberculosis Established

Dr. Allen Kane, medical superintendent of the Municipal Sanatorium at Otisville, N. Y., and a Fellow of the American College of Chest Physicians, has been appointed director of a division of tuberculosis newly established in the New York City Department of Hospitals. Dr. Kane will supervise care of the tuberculous in city hospitals, promote coordination of the tuberculosis service of the department of hospitals with that of private hospitals and perform various other duties directed toward improvement of the service.

First Tuberculosis Sanatorium in Florida

Dr. Jay Arthur Myers, Minneapolis, a Fellow of the American College of Chest Physicians, and president of the National Tuberculosis Association, was the principal speaker when the first state tuberculosis sanatorium in Florida was opened on January 3rd. The sanatorium is a three story brick and concrete structure and located on one of the highest points adjacent to Orlando. It is equipped to accomodate 312 patients but will house more than 400 when the state tuberculosis board has funds to purchase additional equipment.

Tuberculosis Institute

Dr. William Atmar Smith, Charleston, S. C., a Fellow of the American College of Chest Physicians, spoke on the medical and scientific basis of the tuberculosis movement at an institute for health workers at the University of South Carolina, Columbia, March 14-26, held in cooperation with the state tuberculosis association.

SOCIETY NEWS

Dr. Andrew C. Henske, St. Louis, Mo., a Fellow of the American College of Chest Physicians, addressed the Jackson County Medical Society at Carbondale, Illinois, on "Early Symptoms and Diagnosis of Pulmonary Tuberculosis."

Dr. James L. Mudd, St. Louis, Mo., a Fellow of the American College of Chest Physicians, discussed "Surgical Treatment of Pulmonary Tuberculosis" before the Jackson County Medical Society at Carbondale, Illinois.

Dr. James Marr Bisaillon, a Fellow of the American College of Chest Physicians, and Dr. Thomas D. Robertson, Portland, Oregon, addressed the Multnomah County Medical Society, Portland, February 16th, on "Lobar Pneumonia: Some New Aspects, Including Typing."

Dr. Chevalier L. Jackson, Philadelphia, a Fellow of the American College of Chest Physicians, addressed the Columbia Medical Society of Richmond County, Columbia, S. C., February 14th, on "Foreign Body in the Air and Food Passages."

Dr. Louis H. Clerf, a Fellow of the American College of Chest Physicians, and Dr. Howard W. Bradshaw, Philadelphia, addressed the Northampton County Medical Society March 18th on "Pulmonary Infections and Their Treatment."

Postgraduate Course

The medical staff of the Menninger Clinic will conduct its fourth annual Postgraduate Course on *Neuropsychiatry in General Practice,* April 25 to 30, inclusive, at the Menninger Clinic, Topeka, Kansas. The course this year will include a brief introduction to the fields of neurology and psychiatry and a specific application of this knowledge to the large group of cases of psychoneuroses, psychoses and psychogenic and neurological disorders which every psycician meets in his daily practice. Suggestions made by those who took the course last year have been embodied in this year's program in order to make it applicable to the most common practical problems of the physician.

As in previous years, several guest speakers, prominent in the fields of neurology and psychiatry, will appear at the evening sessions of the course.